

The Evolution of Virulence — I

A population of disease-free hosts grows according to the simple ODE

$$\frac{dN}{dt} = [b(N) - d(N)]N$$

where N is population density, $b(N)$ is the density-dependent birth rate and $d(N)$ is the death rate. We assume that either $b(N)$ or $-d(N)$ is a monotonic decreasing function and the other is constant.

Consider a disease that causes lifelong infection (no recovery). Infected individuals die at a rate of $d(N) + \alpha$, where α , the extra mortality caused by the disease, is called the *virulence*. All individuals are born free of the disease. The disease is transmitted between infected and susceptible individuals at rate β , such that the dynamics of susceptible (S) and infected (I) individuals are given by

$$\begin{aligned}\frac{dS}{dt} &= b(N)N - \beta SI - d(N)S \\ \frac{dI}{dt} &= \beta SI - [d(N) + \alpha]I\end{aligned}$$

with $N = S + I$.

The disease increases the rate of mortality because the pathogen is using resources to multiply in the body of the host and because it produces symptoms (such as coughing, bleeding, etc.) which enhance its transmission to other host individuals. Faster replication in the body and more symptoms ensure a higher transmission rate but cause higher mortality as well. We thus assume that β is an increasing function of virulence. Note however that β cannot exceed the contact rate between individuals, hence the function $\beta(\alpha)$ will saturate for large values of α . Different strains of the pathogen differ in their virulence, α , and in the associated transmission rate, $\beta(\alpha)$. We say that a strain is *viable* if it can spread in a disease-free population.

We further assume that once a host is infected with a certain strain cannot be infected by any other strain (no co-infection or superinfection). The dynamics of infection in a population with n different strains is given by one of two models depending on whether density dependence acts in the birth rate (Model 1) or in the death rate (Model 2):

Model 1

$$\frac{dS}{dt} = b(N)N - \sum_{i=1}^n \beta(\alpha_i)I_i S - dS$$
$$\frac{dI_i}{dt} = \beta(\alpha_i)I_i S - [d + \alpha_i]I_i \quad \text{for } i = 1, \dots, n$$

where I_i is the density of hosts infected with strain α_i and $N = S + \sum_{i=1}^n I_i$;

Model 2

$$\frac{dS}{dt} = bN - \sum_{i=1}^n \beta(\alpha_i)I_i S - d(N)S$$
$$\frac{dI_i}{dt} = \beta(\alpha_i)I_i S - [d(N) + \alpha_i]I_i \quad \text{for } i = 1, \dots, n$$

with the same notation as in Model 1.

The aim of this project is to explore the adaptive dynamics of virulence. To this end, it is useful to consider the following questions (you may also add your own):

- (1) Show that in Model 1, different strains of the pathogen cannot coexist in equilibrium. Find the evolutionarily stable strain α^* .
- (2) Suppose that in Model 2, $\beta(\alpha)$ is concave and the viable strains are in an interval (α_m, α_M) with $0 \leq \alpha_m < \alpha_M$. Prove that there is a unique evolutionarily singular strain, which is both convergence stable and evolutionarily stable.
- (3) Show by example that coexistence of two strains is possible in Model 2. Hint: a simple hyperbolic function $\beta(\alpha) = c\alpha / [a + \alpha]$ and a linear function $d(N) = A + BN$ is a good starting point for numerical work.
- (4) Construct a function $\beta(\alpha)$ such that the model exhibits evolutionary branching.
- (5) Explore the coevolution of two strains using isocline plots.